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HEART FAILURE (HJ EISEN, SECTION EDITOR)

Review of Pathophysiology of Cardiogenic Shock and Escalation of Mechanical Circulatory Support Devices

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Abstract

Purpose of Review Cardiogenic shock (CS) is a complex clinical entity that continues to carry a high risk of mortality. The landscape of CS management has changed with the advent of several temporary mechanical circulatory support (MCS) devices designed to provide hemodynamic support. It remains challenging to understand the role of different temporary MCS devices in patients with CS, as many of these patients are critically ill, requiring complex care with multiple MCS device options. Each temporary MCS device can provide different types and levels of hemodynamic support. It is important to understand the risk/benefit profile of each one of them for appropriate device selection in patients with CS.

Recent Findings MCS may be beneficial in CS patients through augmentation of cardiac output with subsequent improvement of systemic perfusion. Selecting the optimal MCS device depends on several variables including the underlying etiology of CS, clinical strategy of MCS use (bridge to recovery, bridge to transplant or durable MCS, or abridge to decision), amount of hemodynamic support needed, associated respiratory failure, and institutional preference. Furthermore, it is even more challenging to determine the appropriate time to escalate from one MCS device to another or combine different MCS devices.

Summary In this review, we discuss the current available data published in the literature on the management of CS and propose a standardized approach for escalation of MCS devices in patients with CS. Shock teams can play an important role to help in hemodynamic-guided management and algorithm-based step-by-step approach in early initiation and escalation of temporary MCS devices at different stages of CS. It is important to define the etiology of CS, and stage of shock and recognize univentricular vs biventricular shock for appropriate device selection and escalation of therapy.

Keywords Cardiogenic shock · Mechanical circulatory support · Shock team · Hemodynamic support · Heart failure

Introduction

Cardiogenic shock (CS) continues to carry a high mortality risk, with an estimated rate of 50% at 6–12 months, despite significant advancements in temporary mechanical circulatory support (MCS) technology over the past two decades [1, 2]. Management of CS remains a significant challenge due to limited randomized-controlled-trial data available to guide its treatment. Mortality is further increased in cases of refractory shock, with reported mortality rates of up to 70% [3]. Pharmacologic therapy is often the initial treatment of choice. However, the use of inotropes and vasopressors is frequently limited by worsening metabolic derangements and imbalance between myocardial oxygen supply and demand.

The landscape of CS management has changed with the advent of several temporary MCS devices designed to provide hemodynamic support. MCS may be beneficial in CS patients through augmentation of cardiac output (CO) with subsequent improvement of systemic perfusion. MCS may also help by decoupling systemic perfusion from myocardial workload and energy expenditure. This is important in all forms of CS but particularly in patients with acute myocardial infarction (AMI)-CS, as MCS can reduce the burden of myocardial ischemia [4].

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Each type of temporary MCS device used for therapeutic escalation from pharmacological therapy carries its own advantages and disadvantages, including technical challenges with insertion, differences in hemodynamic impact, and adverse events. Selecting the optimal MCS device depends on several variables, including the underlying etiology of CS, clinical strategy of MCS use (bridge to recovery, bridge to transplant or durable MCS, or bridge to decision), amount of hemodynamic support needed, associated respiratory failure, and institutional preference. Furthermore, it is even more challenging to determine the appropriate time to escalate from one MCS device to another or combine different MCS devices to better support the haemodynamics and clinical needs of a rapidly deteriorating patient in refractory CS. In this review, we discuss the current available data published in the literature on the management of CS and propose a standardized approach for escalation of MCS devices in patients with CS. This manuscript is a review of the literature and, as such, is exempt from institutional review board oversight.

Cardiogenic Shock Definition Challenge

Historically, there has been a lack of consensus regarding the definition of CS. One of the main obstacles has been the paucity of available data to stratify the different clinical stages of CS. This has created a significant barrier in determining the appropriate selection and use of temporary MCS devices. The most accepted general definition of CS is the state of low CO secondary to cardiac pathology resulting in end-organ dysfunction due to inadequate perfusion. The Society for Cardiovascular Angiography and Interventions (SCAI) recently proposed a new classification that better defines the different stages of CS [5].

The SCAI definition classifies CS into 5 stages:

- Stage A — "At risk," includes patients at risk for CS but not currently experiencing signs and symptoms;
- Stage B — "Beginning," includes patients with clinical evidence of hypotension or tachycardia without hypoperfusion;
- Stage C — "Classic," includes patients with Stage B and evidence of hypoperfusion (requiring intervention such as inotrope, vasopressors or MCS);
- Stage D — "Deteriorating," includes patients who continue to deteriorate despite initial treatment measures and who require multiple vasopressors or addition of MCS; and
- Stage E — "Extremis," includes patients with CS experiencing cardiac arrest being treated with ongoing cardiopulmonary resuscitation and/or extracorporeal membrane oxygenation (ECMO).

Although the new definition of CS has better practical utility, there are few studies that have subsequently validated clinical outcomes, such as survival [3, 6–8]. One major limitation of the new classification system is that it lacks hemodynamic criteria and does not differentiate between univentricular and biventricular shock. This is essential for determining the potential benefit and choice of MCS device.

CS Pathophysiology and MCS Role

In order to determine the type of temporary MCS to use in patients with CS, it is important to first understand the pathophysiology of CS. The overall understanding of the complexity and pathophysiology of CS has evolved [2, 9, 10••]. AMI is the most common etiology of CS and results in derangement of both systolic and diastolic ventricular function, leading to a significant decline in myocardial contractility. Additionally, the mechanical complications associated with AMI can result in acute biventricular dysfunction. This causes a vicious spiral of reduced CO leading to hypotension, subsequently decreasing coronary perfusion, and creating further myocardial impairment. About 30% of the CS cases may include patients with chronic heart failure (HF) and dilated cardiomyopathy presenting in an acute decompensated state. The pathophysiology of HF-CS varies significantly from patients with AMI-CS because of chronic remodeling of the ventricle and neurohormonal changes. However, continuous worsening of CS in both AMI and HF in later stages results in profound vasodilatation from a marked systemic inflammatory response via cytokine release and results in worsening end-organ dysfunction [11].

Hypotension in both AMI and HF-CS results in compensatory vasoconstriction, including venoconstriction, which results in a shift of blood volume from the splanchnic reservoir to the circulatory compartment, resulting in hemodynamic congestion and altering the ventricular-arterial coupling in patients with CS. Hypoperfusion from decreased cardiac output and elevated vascular tone both contributes to the microcirculatory dysfunction from delayed capillary response to the metabolic derangement and results in worsening cellular hypoxia, which eventually results in mitochondrial dysfunction and end-organ dysfunction. This can result in overt inflammation over the following 48 h and may result in marked vasoplegia from increased cytokines (interleukin-1 beta, 6, 7, 8, and 10). It also results in in gluconeogenesis and beta-oxidation of fatty acids; these advanced glycation end-products result in further vasodilatation and are associated with increased mortality. Hence, the multi-organ failure in patients with refractory CS is from both microcirculatory dysfunction and hemodynamic abnormalities (Fig. 1). The gastrointestinal system is often the first organ system involved in CS, from the microcirculatory injury of the intestinal cells resulting in translocation of the intestinal bacteria. Endotoxins and lipopolysaccharide produced by

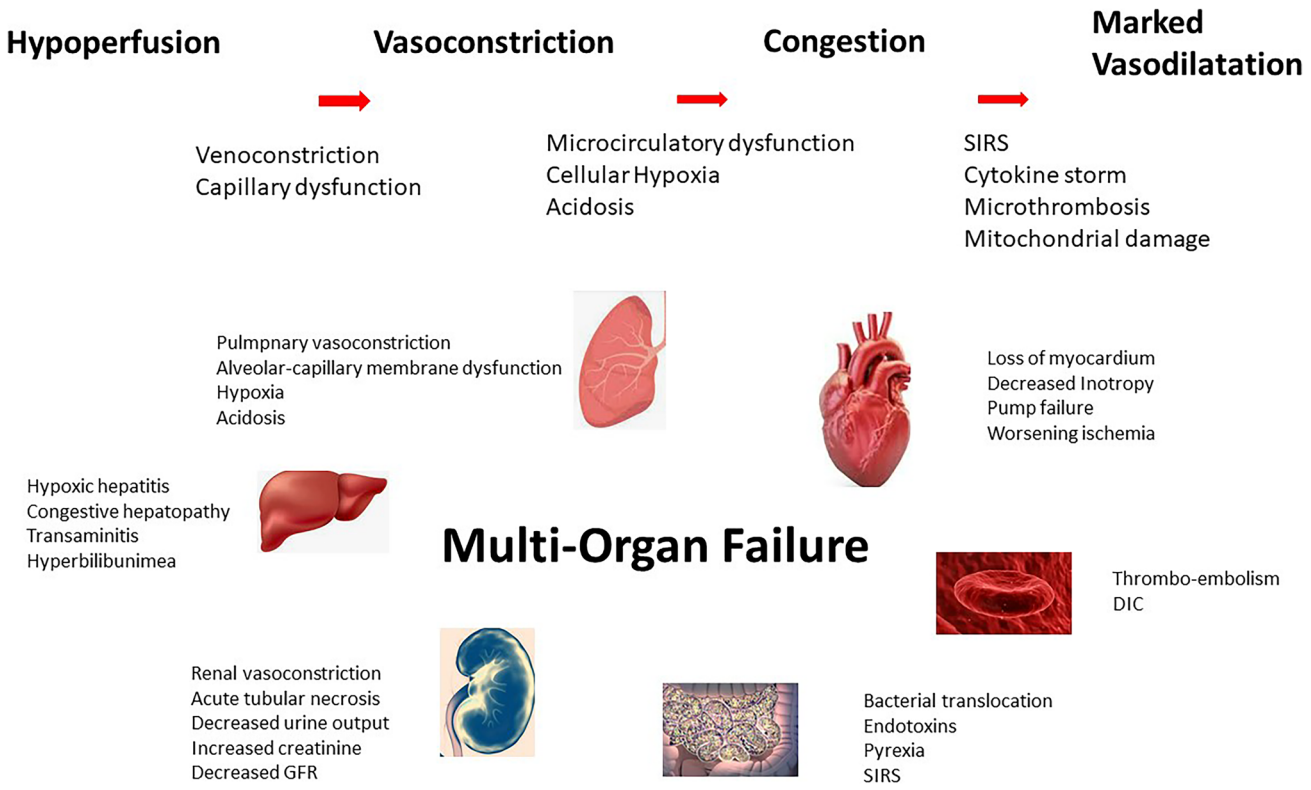

Fig. 1 Pathophysiology of cardiogenic shock

the gram-negative bacteria results in systemic inflammatory response syndrome that results in marked vasoplegia [12].

Therefore, it is very important to recognize and stabilize patients in the early stages of CS to prevent the development of this profound inflammatory state. The goal of escalation to temporary MCS device therapy is to provide early hemodynamic stability and interrupt the spiral of worsening CS (Fig. 2) [13].

MCS Device Types

Each temporary MCS device has its own mechanism of action and can provide different types and levels of hemodynamic support. Each device is also associated with a specific profile of potential complications. It is important to understand the risk/benefit profile of each one in order to recognize its role in the management of various stages of CS. Table 1 summarizes the characteristics of each device.

Left Ventricular Support Devices

Intra-Aortic Balloon Pump

The intra-aortic balloon pump (IABP) is a counterpulsation pump placed percutaneously in the descending aorta. It is one of the most widely used temporary MCS devices, as it is relatively easy to insert, inexpensive, and associated with a good profile of adverse events. The IABP inflates during diastole to improve coronary perfusion, deflates during systole to provide afterload reduction, and can provide 0.5–1 L/min augmentation of CO [14]. The device also helps by reducing left ventricular (LV) filling pressures, which improves interventricular septum positioning and increases the septal contribution to right ventricular (RV) function. In addition, the IABP can augment RV function by increasing flow through the right coronary artery during diastole [15]. The IABP is usually used in the early stages of mild CS and often requires escalation to a more robust MCS device in patients with worsening shock. Prior studies on the use of IABP in AMI-CS failed to demonstrate any survival benefit with its routine use compared to medical therapy [1, 16]. Clinical guidelines have recommended against the routine use of IABP in patients with AMI complicated by CS [17]. However, recent observational studies have highlighted the clinical benefits of the IABP in patients with chronic heart failure with acute decompensation as a bridge to durable MCS such as the left ventricular assist device (LVAD) or heart transplantation [18–20].

Impella

The Impella family of devices (Abiomed, Danvers, MA, USA) includes transvalvular axial flow pumps that can provide

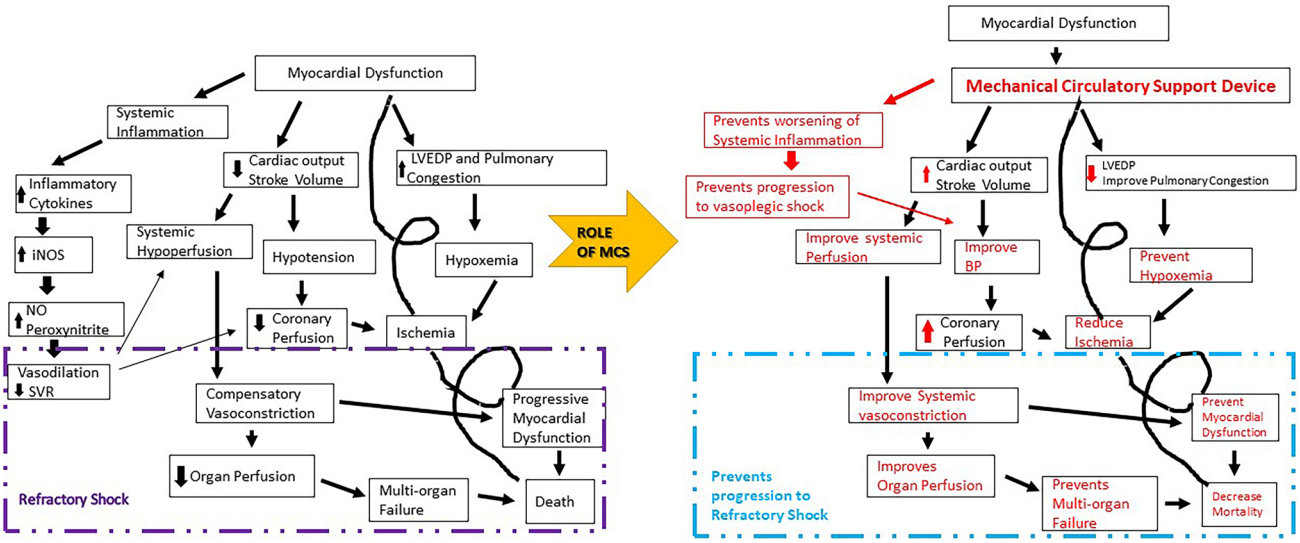

Fig. 2 Role of mechanical circulatory support in improving pathophysiology of cardiogenic shock

hemodynamic support ranging from 2.5 to 6 L/min. The use of Impella has significantly increased in recent years, and it has become one of the commonly used devices for patients with severe CS and LV dysfunction [21, 22]. The newer devices, Impella 5.0 and 5.5, are implanted surgically via the axillary artery or directly into the ascending aorta and can provide flows of up to 6 L/min. Although some of these devices can provide complete LV support with improved hemodynamic effects on myocardial oxygen consumption [21], there are no randomized trials demonstrating a survival benefit in patients with CS [23, 24]. However, retrospective non-randomized clinical data have shown a potential benefit in this patient population [25–28]. Two small randomized controlled trials, ISAR-SHOCK and IMPRESS evaluated the Impella 2.5 and CP devices against IABP in patients with AMI-CS; both failed to show any survival benefit [24, 29].

TandemHeart

TandemHeart (Cardiac Assist, Inc.; Pittsburgh, PA, USA) is a left atrial to femoral artery MCS support device that requires a transseptal atrial cannula for drainage, an arterial return cannula, and an extracorporeal centrifugal flow pump, and can deliver up to 5 L/min. Transseptal puncture is one disadvantage of using this device, and it requires a longer time for implantation. As blood is drawn directly from the left atrium with this device, it results in a significant decrease in pulmonary capillary wedge pressure and LV end-diastolic pressure. However, because the arterial flow is retrograde, it increases the afterload to the left ventricle during systole. This results in no change or a slight decrease in myocardial oxygen consumption [30]. Similar to the Impella, a small-scale study showed that there was no survival benefit of TandemHeart when compared to IABP in the management of AMI-CS [31]. However, the device may be useful in specific cases of CS requiring higher CO support, such as in the presence of mitral regurgitation with severe pulmonary hypertension, RV decompression, or in cases where the Impella device cannot be used, such as in patients with severe aortic stenosis [32, 33]. The TandemHeart provides an additional benefit by allowing the use of an oxygenator that can be spliced into an arterial return circuit in patients with hypoxemia due to severe pulmonary oedema.

Right Ventricular Support Devices

Impella RP

The Impella RP, like other Impella devices, is an axial flow pump that is placed through the femoral vein, rests in the intra-cardiac position, and delivers blood from the inlet area, which sits in the inferior vena cava, to the outlet area, positioned in the proximal pulmonary artery above the pulmonary valve. The use of Impella RP can lead to significant improvement in RV haemodynamics and can provide up to 4 L/min of flow [34]. The device has also been increasingly used for biventricular percutaneous support [35, 36]. However, there are limited data to support its benefit in clinical outcomes. After observing promising results in premarket clinical studies (73% survival), the US Food and Drug Administration issued a warning about a high mortality rate seen in patients with Impella RP in a post-approval study and recommended caution with patient selection and timing of insertion [37].

Table 1 Temporary mechanical circulatory support devices for left and right ventricular cardiogenic shock

Left ventricular MCS								**Right ventricular MCS**		**Biventricular MCS**	
	IABP	Impella 2.5	Impella CP	Impella 5.0	Impella 5.5	TandemHeart	CentriMag	Impella RP	ProtekDuo	CentriMag	ECMO
Mechanism	Pneumatic	Axial Flow	Axial Flow	Axial Flow	Axial Flow	Centrifugal Flow	Centrifugal Flow	Axial Flow	Centrifugal Flow	Centrifugal Flow	Centrifugal Flow
Canula size (in French)	8	13	14	21	21	12–19 Arterial 21 Venous	20–24 Arterial 32–40 Inflow	22 Venous	29/31 Venous	20-24F Arterial 32-40F Inflow	12–19 Arterial 21–25 Venous
Inflow	N/A	LV	LV	LV	LV	LA	LA or LV	IVC	RA	LA or LV (LVAD) RA (RVAD)	RA/IVC/SVC
Outflow	N/A	Aorta	Aorta	Aorta	Aorta	FA	Aorta	PA	PA	Aorta/PA	Aorta/FA/axillary artery
Effect on LV afterload	Reduce (+)	Reduce (++)	Reduce (+++)	Reduce (++++)	Reduce (++++)	Increase	Decrease	-	-	-	Increase
Effect on RV afterload	-	-	-	-	-	-	-	Reduce (+)	Reduce (+)	Reduce (++)	Reduce (++)
Max flow (L/min)	1.0	2.5	3.5	5.0	6.0	5.0	> 5	4.0	5	> 5	> 5
Maximum implant days	Weeks	Days	Days	Weeks	Weeks	Days-Weeks	Weeks	Days	Weeks	Weeks	Weeks
Amount of anticoagulation	Low	Low	Low	Low	Low	High	High	Low	Moderate	Moderate	High
Coronary perfusion	Improves	Improves	Improves	Improves	Improves	-	Improves	-	-	-	-
Hemolysis	Low	Moderate	Moderate	Low	Low	Low	Very Low	Moderate	Low	Low	Low
Risk of limb ischemia	Low	Moderate	Moderate	Low	Low	Mod-High	Low	Moderate	Low	Low	Mod-High

MCS mechanical circulatory support, *IABP* intra-aortic balloon pump, *ECMO* extracorporeal membrane oxygenation, *LA* left atrial, *LV* left ventricular, *RV* right ventricular, *IVC* inferior vena cava, *RVAD* right ventricular assist device, *LVAD* left ventricular assist device

ProtekDuo

ProtekDuo (TandemLife, Pittsburgh, PA, USA), an extracorporeal pump, is a recently introduced percutaneous right ventricular assist device (RVAD) consisting of a percutaneously placed dual-lumen cannula that is inserted through the right internal jugular vein (IJV) to the pulmonary artery. It is used in patients with RV failure and can provide isolated RV support. Similar to the TandemHeart, ProtekDuo can provide up to 5 L/min of flow [38]. In addition, an oxygenator can be added to the system in patients with hypoxemia. A distinct advantage of the ProtekDuo over the Impella RP is the ability for patients to ambulate because it is placed in the IJV [39].

Biventricular Support Devices

Veno-arterial Extracorporeal Membrane Oxygenation

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) can provide more than 6 L/min of hemodynamic support. This platform can be quickly deployed in multiple hospital locations, including the patient's bedside, and provides both respiratory and circulatory support. The most frequently used configuration is peripheral VA-ECMO, which involves the use of venous cannulation that advances a multihole cannula into the right atrium. Blood is drained into an extra-corporeal pump, which is connected to a membrane oxygenator, leading to oxygenation and decarboxylation. Blood is then pumped in a retrograde fashion into the femoral artery. Central surgical configuration via a median sternotomy, used less frequently currently, involves cannulation of the right atrium or pulmonary artery for venous drainage, while the outflow cannula is placed in the ascending aorta. ECMO circuits are increasingly being used in patients with refractory CS. VA-ECMO has also received interest as a possible option for extracorporeal cardiopulmonary resuscitation (ECPR) in patients with cardiac arrest [40].

CentriMag

Surgically implanted centrifugal flow pumps such as the CentriMag pumps (Abbott, St. Paul, MN, USA) are short-term paracorporeal pumps that can be used for LV, RV, or biventricular support in patients with refractory CS and can provide hemodynamic support of up to 10 L/min. An oxygenator can also be placed in patients with severe hypoxemia. It can be used to provide support for up to 6 weeks. However, the need for invasive surgical insertion techniques makes these pumps impractical as an initial device strategy for most patients presenting with acute hemodynamic compromise.

Pulmonary Artery Catheter Role in MCS

Widespread use of temporary MCS has resulted in increased utilization of pulmonary artery catheters (PACs) in order to obtain hemodynamic data for algorithm-based management of patients at different stages of CS [25, 41]. Hemodynamic data can help determine the need for early utilization of temporary MCS devices in patients with inadequate systemic perfusion and worsening respiratory distress [42•]. Previously, the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial had demonstrated no benefit for the use of PACs in patients with acute decompensated HF. However, this study did not include patients with CS [43]. A recent multicenter registry involving the use of a complete PAC hemodynamic profile (right atrial pressure, pulmonary artery systolic and diastolic pressure, pulmonary capillary wedge pressure, and CO) before MCS initiation demonstrated a reduction in mortality in patients of all stages of SCAI CS classification [6]. Complete hemodynamic data can also help differentiate among predominantly LV, RV, or biventricular shock [44, 45].

Determining the CO in patients with CS is essential. In a post hoc analysis of the SHOCK trial, cardiac power output (CPO) was the strongest predictor of 30-day mortality in patients with CS [46]. This result suggests that PAC can help in early initiation or escalation of temporary MCS devices. If the hemodynamic profile continues to worsen on the initial MCS device, it may be advantageous to rapidly escalate therapy to a device that can provide more robust hemodynamic support [30]. PACs also provide the advantage of continuous monitoring using real-time data to understand the effectiveness of pharmacologic and MCS and pharmacologic strategies.

Although CPO is an important hemodynamic marker, it does not help in delineating RV or biventricular shock from LV shock [47]. PAC plays an important role in identifying indices and markers of RV dysfunction and shock, such as the calculation of RA pressure to pulmonary capillary wedge pressure and pulmonary artery pulsatility index, which is defined as the ratio of pulmonary artery pulse pressure to right atrial pressure. Both are important hemodynamic markers for RV failure. RV stroke work is another important hemodynamic parameter that helps in recognizing RV dysfunction [48, 49]. Many patients who are under-supported by IABP or LV support devices have underlying RV failure and carry a high mortality risk if this inadequate support is not recognized [19]. RV dysfunction is common and was seen in about 38% of the patients with AMI-CS in the SHOCK trial [49].

Clinical, Laboratory, and Imaging Data

In the absence of randomized data, clinical judgment regarding escalation should be exercised by close monitoring of clinical and hemometabolic data. It is important to monitor daily clinical and laboratory markers to recognize signs of end-organ dysfunction. These markers can help not only in the early identification of patients who are undersupported or those with underlying RV failure, but also in the recognition of complications associated with MCS devices. Table 2 lists the important laboratory and clinical markers used for monitoring and managing CS.

Echocardiography is indispensable in identifying the etiology of CS and understanding haemodynamics. Imaging also plays an important role in identifying various complications that may require immediate attention. Mechanical complications of AMI, such as papillary muscle rupture or ventricular septal defect, cardiac tamponade, and valvular abnormalities, are frequently associated with CS, all of which can be recognized with an echocardiogram [32, 50, 51]. Echocardiographic imaging is also important in assessing adequate positioning of different temporary MCS devices in the management of CS [52].

Cardiogenic Shock Team Model

Despite the wide availability of multiple MCS devices for the management of CS, mortality remains high. Significant variation in the management of CS between clinicians and late referral of these patients to advanced shock centers may contribute to this high mortality [53, 54]. Recently, increased emphasis has been placed on the development of a multidisciplinary "shock team" in order to reduce this variability and develop a standardized approach to the care of patients with CS [25, 41]. This multidisciplinary team, consisting of interventional cardiology, advanced HF, critical care, and cardiac surgery, can help to facilitate early recognition and timely escalation of appropriate MCS devices in patients with CS and has led to improved clinical outcomes (Fig. 3) [55, 56]. This shared decision-making in the management of these critically ill patients can potentially

Table 2 Laboratory and clinical markers to monitor end-organ function

Type of organ dysfunction	Laboratory and clinical markers	Comments
Acute renal failure	Daily monitoring of Creatinine and GFR Hourly urine output	Common manifestation of CS due to hypoperfusion and volume overload Worsening CVP: Renal venous congestion Worsening AKI in patients with LV MCS support may suggest need for RV support
Acute liver injury	Daily liver function Tests	Worsening transaminitis and elevated bilirubin suggestive of RV failure and venous hypertension Raise suspicion for Biventricular shock
Peripheral/tissue hypoperfusion	Lactic acid levels	Worsening lactic acidosis - Suggestive of inadequate hemodynamic support Patients on IABP/Impella CP may need escalation to higher support devices such as Impella 5.0/5.5 Look for signs of RV shock/Biventricular shock Look for acute limb ischemia in patients with large bore access with no peripheral pulses
Respiratory failure	Daily chest x-ray Arterial blood gas – hypoxia (Pao2: Fio2)	Consider MCS devices with oxygenator for patients with worsening hypoxemia Consider LV venting in patients on VA-ECMO support
Brain	Check for daily neurological signs off sedation Lactate levels	Refractory shock can result in cerebral hypoperfusion – check lactate levels and signs of other hypoperfusion Patient on MCS – Look for signs of stroke; consider CT Head
Hematology	Daily hemoglobin/hematocrit Plasma free hemoglobin in patients with hemolysis Platelet levels	Worsening anemia – look for signs of bleeding in patients with MCS requiring anticoagulation Signs of hemolysis- worsening plasma free hemoglobin, elevated LDH levels, low haptoglobin Thrombocytopenia – common with MCS devices. Rule out HIT

GFR glomerular filtration rate, *CS* cardiogenic shock, *CVP* Central venous pressure, *AKI* acute kidney injury, *RV* right ventricular, *LV* left ventricular, *IABP* intra-aortic balloon pump, *VA-ECMO* veno-arterial extracorporeal membrane oxygenator, *LDH* lactate dehydrogenase, *HIT* heparin-induced thrombocytopenia

improve outcomes in a field where significant high-quality evidence-based guideline recommendations are still limited. Use of a shock team also provides the opportunity to effectively collect high-quality data, with more rigorous evaluation of clinical outcomes and the impact of interventions with these devices. Additionally, early engagement of advanced HF in patients with refractory CS helps in early identification of patients without left ventricular recovery who may require durable LVAD and heart transplantation.

MCS Escalation in Cardiogenic Shock

Currently, there are limited randomized-controlled-trial data on the use of the above-mentioned MCS devices. Protocols for utilization of MCS treatment in patients with CS are not standardized and are specific to the operators and institutions based on diverse experience levels with the different types of devices. The optimal timing of utilization and escalation of MCS devices has not been well-established.

As shown in Fig. 4a, the first step in the management of CS is to determine the possible etiology of CS (AMI vs. HF). Patients with AMI-CS should be immediately taken to the cardiac catheterization laboratory for early revascularization and hemodynamic stabilization. It is also important to rapidly identify the SCAI staging for any type of CS by recognizing clinical severity using laboratory, hemodynamic, and imaging data [6, 41, 45]. At this point, it is essential to expedite a shock-team discussion in order to determine the role of temporary MCS support. CS is a continuum of hemodynamic instability and involves both hypotension and hypoperfusion. Patients with isolated hypoperfusion have a higher mortality risk than hypotensive patients with normal perfusion [57].

Figures 4b and 5 demonstrate the possible hemodynamic deficit and role of different MCS devices at different stages of shock. Additionally, the body surface area of the patient should be considered at the time of initial MCS selection. Patients with high body surface area may require MCS devices that provide higher hemodynamic support. Before MCS device selection, it is essential to recognize the difference between univentricular vs. biventricular shock and concomitant respiratory failure for appropriate temporary MCS selection.

Univentricular Shock

LV Dominant Shock

Once it is recognized that the patient is in CS, the next step involves performing a right heart catheterization to determine the degree of cardiac output deficit (Fig. 5). A recent study showed that CS patients who underwent PAC with complete hemodynamic data prior to MCS initiation had better survival [6]. These differences were more pronounced in patients with refractory CS (Stage D-E). IABP and Impella CP are the most frequently used devices in early-stage C CS both for patients with AMI-CS and HF-CS with CPO from 0.6 to 1 W [45]. If the patient continues to deteriorate, the most common sequence in escalation involves transitioning from IABP/Impella CP support to an Impella 5.0/5.5 [21, 58, 59]. Additionally, Impella 5.0/5.5 may also be used as the initial MCS device in patients with CPO < 0.6 W to provide adequate hemodynamic support [60].

Impella 5.0 and 5.5 devices are being increasingly used in patients with AMI-CS and post-cardiotomy CS and as a bridge to decision in HF patients, as these devices provide complete hemodynamic support with fewer serious complications in comparison with ECMO [26, 27, 61]. In a meta-analysis performed by Batsides et al., the use of Impella 5.0 was associated with favorable clinical outcomes, with overall 1-year survival of 58.4% in patients with different etiologies of CS [21].

In special circumstances of predominant LV shock where Impella devices may be contraindicated, a TandemHeart can be used. Some of these scenarios include the presence of a prosthetic aortic valve, aortic regurgitation, or intraventricular thrombus (for which Impella devices are contraindicated). TandemHeart may also be used in patients with predominant LV shock complicated by respiratory failure, as it allows for the use of an oxygenator.

RV Dominant Shock

MCS devices can also be used early in patients with CS due to worsening RV failure and RV myocardial infarction. PAC is specifically useful for identifying these patients, as they predominantly have normal LV filling pressure [50, 62]. Figure 4b shows the stepwise management of RV or biventricular shock. Most of these patients are initially treated with medical therapy by reducing preload with continuous diuresis and by using inotropes and pulmonary vasodilators for contractility and reduction of RV afterload. However, if the patient continues to deteriorate, a temporary MCS device may be required. Impella RP and ProtekDuo are the currently available devices for direct RV support. In comparison to Impella RP, ProtekDuo provides the additional advantage of utilizing an oxygenator in patients with concomitant respiratory failure and hypoxemia. VA-ECMO can also be used for predominant RV shock; however, because of the increased risk of complications with VA-ECMO, it should be restricted for biventricular shock patients. CentriMag can also be used for those who require longer duration of RVAD support as a possible bridge to recovery or advanced therapies.

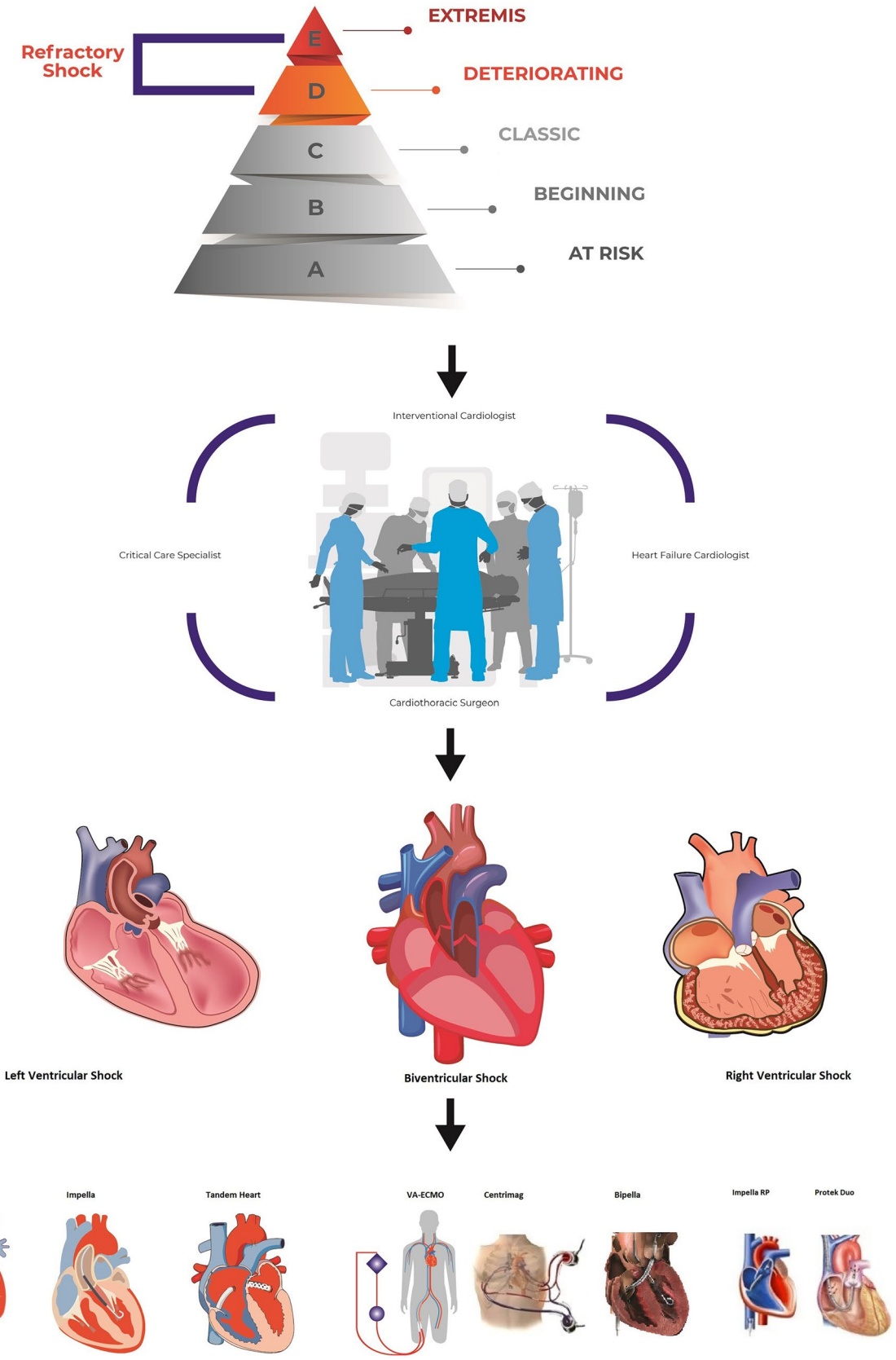

Fig. 3 Proposed algorithm to standardized approach of mechanical circulatory support escalation in different types of cardiogenic shock

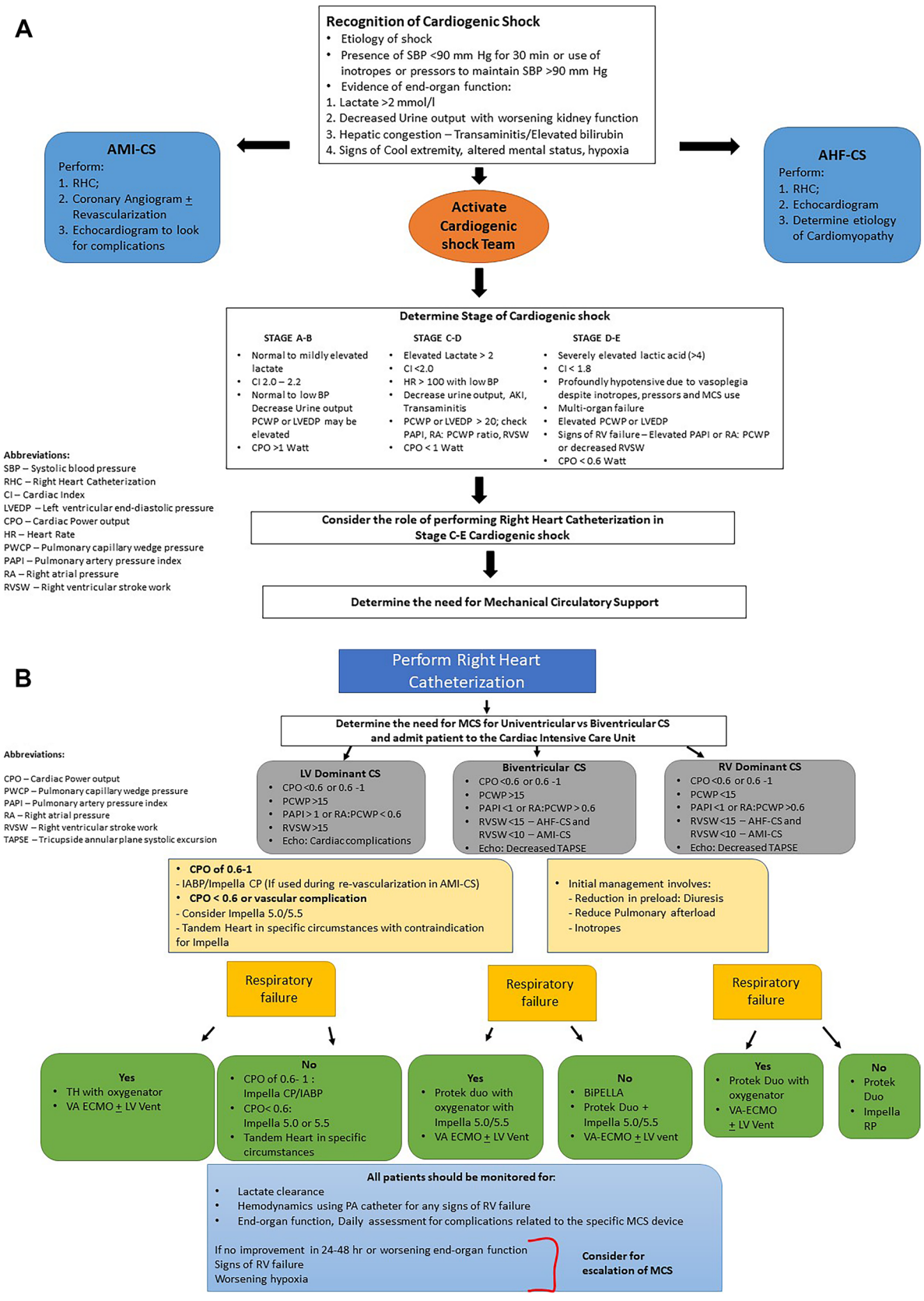
A
Recognition of Cardiogenic Shock
• Etiology of shock
• Presence of SBP <90 mm Hg for 30 min or use of inotropes or pressors to maintain SBP >90 mm Hg
• Evidence of end-organ function:
1. Lactate >2 mmol/l
2. Decreased Urine output with worsening kidney function
3. Hepatic congestion – Transaminitis/Elevated bilirubin
4. Signs of Cool extremity, altered mental status, hypoxia
AMI-CS
Perform:
1. RHC;
2. Coronary Angiogram ± Revascularization
3. Echocardiogram to look for complications
AHF-CS
Perform:
1. RHC;
2. Echocardiogram
3. Determine etiology of Cardiomyopathy
Activate Cardiogenic shock Team
Determine Stage of Cardiogenic shock
STAGE A-B
• Normal to mildly elevated lactate
• CI 2.0 – 2.2
• Normal to low BP Decrease Urine output PCWP or LVEDP may be elevated
• CPO >1 Watt
STAGE C-D
• Elevated Lactate > 2
• CI <2.0
• HR > 100 with low BP
• Decrease urine output, AKI, Transaminitis
• PCWP or LVEDP > 20; check PAPI, RA: PCWP ratio, RVSW
• CPO < 1 Watt
STAGE D-E
• Severely elevated lactic acid (>4)
• CI < 1.8
• Profoundly hypotensive due to vasoplegia despite inotropes, pressors and MCS use
• Multi-organ failure
• Elevated PCWP or LVEDP
• Signs of RV failure – Elevated PAPI or RA: PCWP or decreased RVSW
• CPO < 0.6 Watt
Abbreviations:
SBP – Systolic blood pressure
RHC – Right Heart Catheterization
CI – Cardiac Index
LVEDP – Left ventricular end-diastolic pressure
CPO – Cardiac Power output
HR – Heart Rate
PWCP – Pulmonary capillary wedge pressure
PAPI – Pulmonary artery pressure index
RA – Right atrial pressure
RVSW – Right ventricular stroke work
Consider the role of performing Right Heart Catheterization in Stage C-E Cardiogenic shock
Determine the need for Mechanical Circulatory Support
B
Perform Right Heart Catheterization
Determine the need for MCS for Univentricular vs Biventricular CS and admit patient to the Cardiac Intensive Care Unit
Abbreviations:
CPO – Cardiac Power output
PWCP – Pulmonary capillary wedge pressure
PAPI – Pulmonary artery pressure index
RA – Right atrial pressure
RVSW – Right ventricular stroke work
TAPSE – Tricupside annular plane systolic excursion
LV Dominant CS
• CPO <0.6 or 0.6 -1
• PCWP >15
• PAPI > 1 or RA:PCWP < 0.6
• RVSW >15
• Echo: Cardiac complications
Biventricular CS
• CPO <0.6 or 0.6 -1
• PCWP >15
• PAPI <1 or RA:PCWP > 0.6
• RVSW <15 – AHF-CS and RVSW <10 – AMI-CS
• Echo: Decreased TAPSE
RV Dominant CS
• CPO <0.6 or 0.6 -1
• PCWP <15
• PAPI <1 or RA:PCWP >0.6
• RVSW <15 – AHF-CS and RVSW <10 – AMI-CS
• Echo: Decreased TAPSE
• CPO of 0.6-1
- IABP/Impella CP (If used during re-vascularization in AMI-CS)
• CPO < 0.6 or vascular complication
- Consider Impella 5.0/5.5
- Tandem Heart in specific circumstances with contraindication for Impella
• Initial management involves:
- Reduction in preload: Diuresis
- Reduce Pulmonary afterload
- Inotropes
Respiratory failure
Respiratory failure
Respiratory failure
Yes
• TH with oxygenator
• VA ECMO ± LV Vent
No
• CPO of 0.6- 1 : Impella CP/IABP
• CPO< 0.6: Impella 5.0 or 5.5
• Tandem Heart in specific circumstances
Yes
• Protek duo with oxygenator with Impella 5.0/5.5
• VA ECMO ± LV Vent
No
• BiPELLA
• Protek Duo + Impella 5.0/5.5
• VA-ECMO ± LV vent
Yes
• Protek Duo with oxygenator
• VA-ECMO ± LV Vent
No
• Protek Duo
• Impella RP
All patients should be monitored for:
• Lactate clearance
• Hemodynamics using PA catheter for any signs of RV failure
• End-organ function, Daily assessment for complications related to the specific MCS device
If no improvement in 24-48 hr or worsening end-organ function
Signs of RV failure
Worsening hypoxia
Consider for escalation of MCS

Fig. 4 A Recognition and identification of the type and stage of cardiogenic shock **B** Recognition of univentricular vs. biventricular shock for appropriate mechanical circulatory support selection

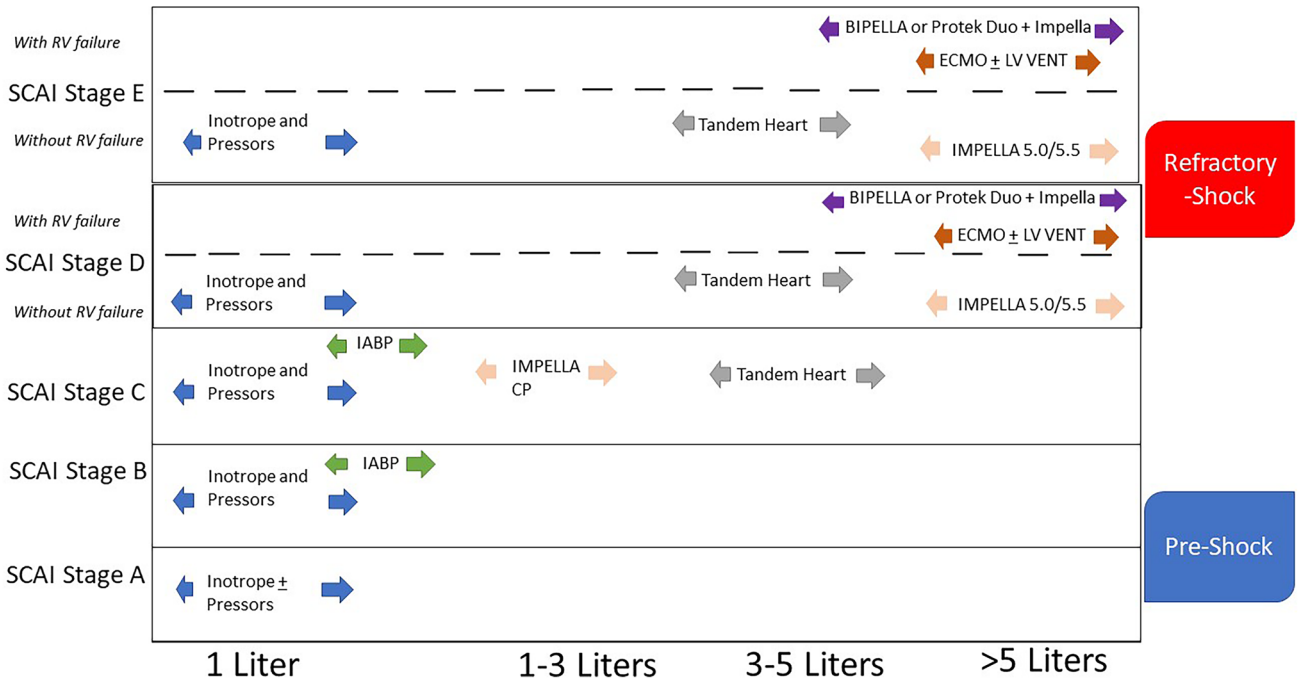

Fig. 5 Medical therapy and available mechanical circulatory support devices for different stages of cardiogenic shock

Biventricular Shock

The most challenging patients are those with biventricular shock who present in advanced stages of refractory CS. These patients require urgent decision-making and escalation of MCS therapy within hours [63]. Early consideration should be given to adding a percutaneous RVAD such as Impella RP or ProtekDuo in patients with LV shock who continue to deteriorate despite adequate LV MCS device support and evidence of RV failure [45, 49]. Many patients may have concomitant respiratory failure and are often escalated directly to VA-ECMO [64]. However, VA-ECMO use is associated with an increased LV afterload and can result in worsening of LV end-diastolic pressure, acute pulmonary oedema, decreased coronary perfusion, and worsening valvular regurgitation. There are emerging data suggesting that using LV venting strategies along with ECMO is associated with improved survival [65]. Table 3 shows different types of LV venting strategies that can be used. Recently, the use of ECPELLA (combination of VA-ECMO and Impella) has gained significant interest, as it can provide active LV unloading and has been shown to have better survival than VA-ECMO alone [66, 67]. Another option for patients with biventricular failure includes using right- and left-sided Impella devices, known as the BIPELLA strategy, or using an Impella device with a ProtekDuo [50]. However, patients requiring more than one device are at significantly increased risk of complications and worse outcomes. Use of multiple devices is also associated with significant economic impact with high hospitalization cost, and hence, it is important to recognize the advantages and disadvantages prior to utilization of multiple devices [63].

Complications Associated with MCS

Despite its hemodynamic benefits, each temporary MCS device is associated with an increased risk of device-related complications. These risks are significantly increased in patients with refractory CS requiring escalation, as many of these patients are already on vasopressors, anticoagulants, or antiplatelets, or on other devices. Many of the MCS devices used for refractory CS require large-bore vascular access and are associated with increased risk of limb ischemia, bleeding, and stroke. It is important to understand these risk factors during device selection, as larger-bore MCS such as ECMO or Impella are associated with a higher risk of complications in comparison with the IABP [68–70]. To decrease the risk of vascular complications, a careful vascular assessment using imaging should be performed to evaluate for vascular size. Strategies such as an antegrade distal perfusion catheter may be necessary to reduce the risk of limb ischemia. Additional strategies to reduce complications include monitoring for signs of hemolysis, confirmation of device placement on a regular basis, and performing daily extensive neurologic examinations to identify signs of a stroke, which can be particularly challenging in unconscious patients. It is crucial to adopt clinical practice guidelines for management of complications associated with MCS as a part

Table 3 Strategies for Left ventricular venting in patients with ECMO

Strategies for LV venting	Advantages	Disadvantages
Passive unloading		
Inotropes	Usually, first line Non-invasive	Increases risk of arrhythmia Increases myocardial oxygen demand
IABP	Reduces afterload Promote aortic valve opening Decreased risk of aortic thrombosis	Increases risk of cerebral and spinal ischemia Vascular complications Probably not very effective
Atrial Septostomy	Left atrial decompression can help provide high flow	Transseptal puncture Increase risk of complications Cannot regulate flow
Active unloading		
ECPELLA (ECMO with Impella)	Decreases LV distension Improves filling pressure and decrease myocardial oxygen demand	Vascular complications Increased hemolysis
Surgical Decompression (Direct canulation of the left atrium or left ventricle)	High flow capacity and can regulate flow	Requires surgery Limited durability Catheter clotting

ECMO extracorporeal membrane oxygenation, *IABP* intra-aortic balloon pump, *ECPELLA* ECMO device with an Impella

of the algorithm for management of CS with MCS devices to avoid significant complications.

Conclusion

CS continues to carry high short-term mortality with a lack of standardization of treatment options at different stages of shock. Understanding the role of temporary MCS in patients with CS remains challenging, as many of these patients are critically ill and require complex care with multiple MCS device options. Shock teams can play an important role in hemodynamic-guided management and algorithm-based step-by-step approach in early initiation and escalation of temporary MCS devices at different stages of CS. It is important to define the etiology and stage of CS and to recognize univentricular vs. biventricular shock for appropriate device selection and escalation of therapy. Despite the increasing prevalence of CS, there are limited data available to better understand the pathophysiology, therapeutic options, and role of different temporary MCS devices. Collaborative international effort is required to conduct clinical trials, develop shock networks, and create large, multicenter registries that can generate better-quality research in this dynamic and evolving field.

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Compliance with Ethical Standards

Ethics Approval This manuscript is a review of the literature and, as such, is exempt from institutional review board oversight.

Conflict of Interest Hayder Hashim reports serving as a consultant for and receiving speaker fees from Abbott, Cardiovascular Systems, Inc., and Boston Scientific. Samer S. Najjar reports receiving grant support from Abbott and Medtronic and receiving support for attending meetings from Abbott. Ron Waksman reports serving on the advisory boards of Abbott Vascular, Boston Scientific, Medtronic, Philips IGT, and Pi-Cardia Ltd.; being a consultant for Abbott Vascular, Biotronik, Boston Scientific, Cordis, Medtronic, Philips IGT, Pi-Cardia Ltd., Swiss Interventional Systems/SIS Medical AG, Transmural Systems Inc., and Venous MedTech; receiving institutional grant support from Amgen, Biotronik, Boston Scientific, Chiesi, Medtronic, and Philips IGT; and being an investor in MedAlliance and Transmural Systems Inc. All other authors have no competing interests to declare that are relevant to the content of this article.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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